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Airway pressure release ventilation

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Learning objectives

By reading this article, you should be able to:

- Describe the physiological rationale for airway pressure release ventilation (APRV).
- State the risks, benefits, indications for and potential contraindications to APRV.
- Outline the evidence supporting the use of APRV.
- Explain how to initiate, titrate, troubleshoot and wean a patient from APRV.

Key points

- Airway pressure release ventilation (APRV) is an open-lung mode of invasive mechanical ventilation mode, in which spontaneous breathing is encouraged.
- APRV uses longer inspiratory times; this results in increased mean airway pressures, which aim to improve oxygenation.
- Brief releases at a lower pressure facilitate carbon dioxide clearance.
- The terminology and methods of initiation, titration, and weaning are distinct from other modes of mechanical ventilation.
- The use of APRV is increasing in the UK despite a current paucity of high-quality evidence.

Airway pressure release ventilation (APRV) is a pressure-controlled mode of ventilation that delivers an almost continuous positive pressure with intermittent, time-cycled, short releases at a lower pressure. Spontaneous ventilation is encouraged, and the relatively increased mean airway pressures allow 'open-lung' ventilation. APRV was first described in 1987 by Stock and colleagues, who demonstrated that arterial oxygenation and carbon dioxide clearance were improved using APRV compared with intermittent positive-pressure ventilation (IPPV) in 10 anaesthetised dogs with normal lungs.[1] Following this original description, APRV has evolved and its use is increasing, particularly in the management of patients with severe acute respiratory failure (SARF) and acute respiratory distress syndrome (ARDS). The fact that there is no universally agreed definition of APRV can make discussion of the topic problematic. This article concentrates on the concept of personalised APRV developed by Habashi.[2] We explain the physiological rationale, risks, benefits and evidence base for APRV, with practical tips on how to initiate, titrate and wean a patient from APRV.

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Nomenclature

The nomenclature used to describe APRV is different to that used in conventional ventilation (Fig. 1). P-high describes the highest level of pressure applied to the respiratory system, and T-high describes the time in seconds spent at this pressure. P-high is distinct from the terms P-Insp and inspiratory pressure that are used to describe conventional mechanical ventilation. This is to convey the prolonged duration spent at

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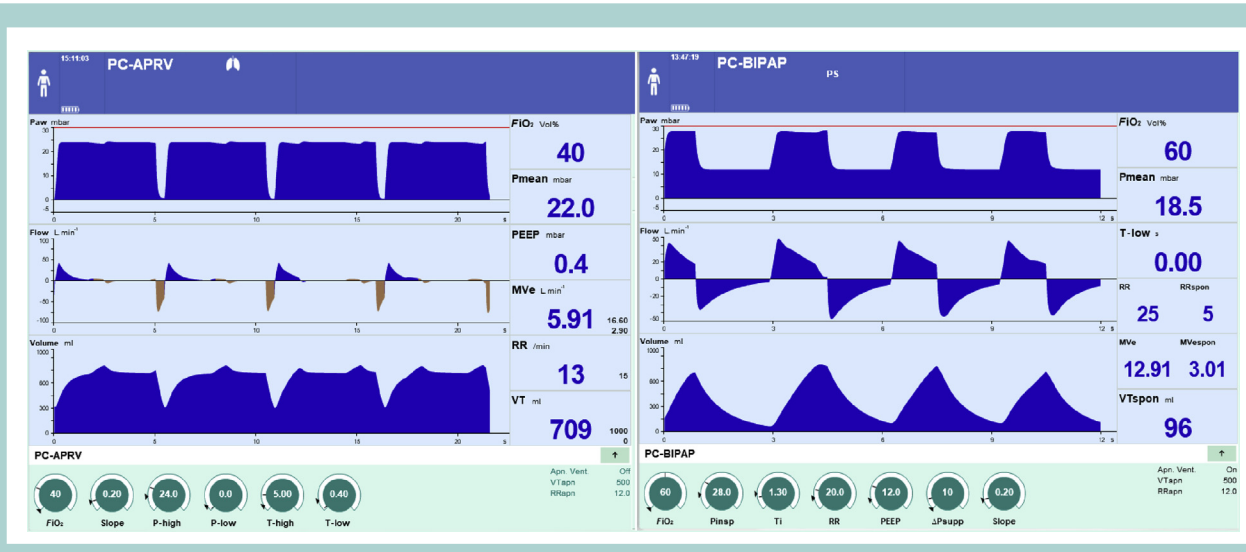

Fig 1 Ventilator screenshots: APRV compared with pressure-controlled IPPV (PC IPPV). BIPAP, Bi-level positive airways pressure.

this pressure, which accounts for the majority of the respiratory cycle.

P-low is the lowest pressure applied by the ventilator to the respiratory system, and T-low indicates the time in seconds spent at this pressure. P-low is normally set at zero cmH_2O, but T-low is short and titrated, such that intrathoracic pressure never reaches atmospheric pressure. This concept is explained further in the section on initiating and titrating APRV.

Physiological rationale for the use of APRV

By maintaining a prolonged high pressure (P-high), APRV maximises the recruitment of available lung tissue and therefore improves oxygenation. This is an example of the 'open-lung' approach to invasive ventilation. Short and relatively infrequent periods at a lower pressure (P-low) facilitate carbon dioxide clearance (see Fig. 1). In effect, APRV is similar to an almost constant recruitment manoeuvre. This is in contrast to conventional invasive ventilation, in which a briefer period of recruitment is used followed by PEEP to prevent alveolar collapse.

Stress and strain are concepts borrowed from mechanical engineering that are increasingly being used to describe lung dynamics in the context of mechanical ventilation.[3] These ideas can be useful when considering the differences between conventional ventilation and APRV. *Stress* is defined as the applied force (encompassing pressure and area change) exerted on the lungs during mechanical ventilation. *Strain* is the resultant deformation of the lungs, expressed as the ratio between end-inspiratory lung volume and end-expiratory lung volume.

The amounts of stress and strain are markedly affected by starting lung volume. Stress and strain are reduced when ventilating a lung that is homogeneous and fully recruited compared with ventilating a minimally recruited, collapsed lung, even if the tidal volume achieved is the same. The ideal technique for ventilation is one that recruits all available alveoli, inflates the lung to the optimal point on its compliance curve and maintains an adequate end expiratory volume. These factors minimise dynamic strain.

In conventional positive-pressure ventilation in patients with lung injury, tidal volumes are restricted to minimise lung stress and incremental amounts of PEEP are used in an attempt to maintain alveolar recruitment during expiration. It is often difficult to determine the optimal PEEP to prevent derecruitment for each patient. When using APRV, prolonged time at an increased mean airway pressure maximises alveolar recruitment by providing a longer time to achieve homogeneous ventilation and gas distribution between lung units that have differing compliances. The dramatically prolonged time at an increased pressure increases the end-expiratory lung volume and therefore reduces dynamic strain.

In contrast to conventional IPPV, derecruitment is prevented by titrating the duration of T-low to alter the expiratory flow characteristics. The flow characteristics themselves are determined by the lung mechanics of each individual patient. These concepts are detailed in the section on initiating and titrating APRV.

During APRV, the patient is encouraged to breathe spontaneously throughout the respiratory cycle. This facilitates gas exchange, maximises continued recruitment of lung tissue, promotes venous return to the heart and prevents wasting of respiratory muscles. In a passive system, non-dependent lung units are recruited first, followed by dependent units. This risks over-distension of the non-dependent units by unnecessarily high pressures. Conversely, spontaneous ventilation in an active system recruits dependent lung units first through pleural pressure change.[2] The method of setting the duration of T-low based on expiratory flow characteristics and the promotion of spontaneous ventilation fundamentally differentiates APRV from extreme inverse ratio mandatory ventilation.

Benefits and risks of APRV

Respiratory

Much research has been performed to identify techniques that may attenuate ventilator-induced lung injury. The harmful effects of volutrauma, barotrauma, atelectrauma and biotrauma in initiating and worsening lung injury have been well described.[4] The sustained high mean airway pressure of

APRV aims to promote alveolar recruitment, improve lung homogeneity and increase functional residual capacity whilst maintaining the benefits associated with spontaneous breathing.[5] During APRV, lung units are kept open more consistently. This theoretically reduces the cyclical opening and collapse of atelectatic but recruitable units, thereby minimising lung injury.

Critics of APRV are wary of the potential for aggravating lung injury. They argue that spontaneous breathing during T-high can cause high local transpulmonary pressures and tachypnoea, especially in the context of heterogeneous lung disease, which in turn may increase the risk of 'patient self-inflicted lung injury'. They also warn that occult atelectrauma still occurs with APRV, as T-low times longer than 0.2 s could still result in collapse of injured alveoli, and many ventilators are unable to provide T-low times this short.[6] 'Lung protective ventilation' in ARDS aims for a tidal volume of $\leq$6 ml kg^{-1} ideal body weight and limits plateau pressures to 30 cmH_20 and is a strategy that has been widely accepted and adopted in the critical care community since the publication of the landmark ARDSnet trial of 2000.[7] In practice plateau pressure may be difficult to measure and therefore many clinicians target a peak inspiratory pressure $\leq$30 cmH_20 in these patients. Efforts to accurately measure and target tidal volumes are problematic with APRV, as is ensuring that the peak pressure remains below 30 cmH_2O.[5] However, many of these concerns are based on findings from computer modelling, and it remains a problem to quantify the degree of occult atelectrauma *in vivo*.

Cardiovascular

Increased intrathoracic pressure as a result of mechanical ventilation has many effects on the heart, both positive and negative.[8] The negative effects are well known: decreased venous return to the right side of the heart, increased afterload and increased pulmonary vascular resistance (which can be catastrophic in patients with right heart failure). However, the positive effects are often neglected: high intrathoracic pressure decreases the transmural left ventricular pressure, reducing the work of contraction and increasing cardiac output. In the context of hypoxaemia, a mode of mechanical ventilation that improves arterial oxygenation will improve myocardial oxygen delivery, myocardial function and cardiac output. As APRV is a spontaneous breathing mode, in addition to the benefits of spontaneous ventilation, reduced doses of sedative drugs can often be used, with subsequent reduction of requirement for vasoactive drugs and improvement in haemodynamic state.[6,9]

These multiple complex cardiorespiratory interactions make it difficult to predict which patients will have an adverse cardiovascular response to APRV. It is our approach to offer a trial of APRV to almost all patients with severe respiratory failure [Pa_{O_2}/FI_{O_2} (PF) ratio <100 mmHg] who we think may benefit. Echocardiographic assessment of left and right ventricular functions before and after initiation of APRV is very useful. In the face of arterial hypotension on initiation, we usually administer a 250–500 ml bolus of i.v. fluids. This is usually sufficient to restore cardiac output. Echocardiography should be used to guide the management of persisting hypotension. Whilst it is important to always choose the most appropriate mode of ventilation for each patient, and APRV is not advocated for the profoundly hypovolaemic under-resuscitated patient, its use in resuscitated trauma patients has been well described.[2,10]

Evidence base for APRV

Studies in animals

There have been multiple experiments predominantly using porcine or rodent animal models to investigate whether APRV confers any advantage or harm compared with conventional ventilation. In animal models, APRV improves arterial oxygenation, increases ventilation in dependent areas of lung, reduces inflammatory cytokine production, and can prevent the development of ARDS.[11–15]

One of the most striking animal experiments was in pigs with induced sepsis, randomised to receive APRV or 'ARDSnet protocol' low tidal volume (LTV) ventilation.[15] APRV was commenced 1 h after the septic stimulus. LTV ventilation (tidal volume 6 ml kg^{-1}) was started in the second group when the pigs met the criteria for mild ARDS (i.e. PF ratio <300 mmHg). In a third 'sham' group with no physiological stimulus for sepsis, the pigs' lungs were ventilated at 10 ml kg^{-1} for the duration of the experiment. Pigs in the LTV group had reduced concentrations of surfactant protein A, poorer histological appearance and inferior gas exchange compared with the APRV group. At post-mortem histological examination, the lung tissue in the APRV group was normal, pink and homogeneously well inflated, whereas the lungs from LTV animals were predominantly atelectatic with heterogeneous parenchymal inflammation (Fig. 2).[15] Normal lung architecture was preserved in the APRV group, whereas in the LTV group there were congested capillaries, fibrous exudates, intra-alveolar haemorrhage and leucocytic infiltrates consistent with lung injury. The authors concluded that APRV might have a role in the prevention of ARDS.

Another similar trial examined respiratory dynamics in a porcine model.[16] Despite increased plateau pressures and tidal volumes in the APRV group, differences in transpulmonary pressures were not statistically significant, reflecting significantly lower pleural pressures in the LTV group. Lung, chest wall and respiratory system elastance were all increased in the LTV group.

Studies in humans

It is important to note that many trials of APRV were conducted before the ARDSNet trial, which demonstrated a reduction in mortality with lower tidal volume ventilation compared with higher tidal volumes; but thus, did not compare APRV to current best practice for mechanical ventilation.[7] Therefore, the historical evidence base must be interpreted with caution. Supplementary Table S1 outlines human trials comparing APRV with LTV over the past decade.

A large retrospective systematic review of 66,199 patients in North American trauma centres showed that early use of APRV in a single centre was associated with lower incidences of both ARDS (1.3% *vs* 14.0%) and mortality (3.9% *vs* 14.1%) than conventional ventilation in other centres.[10] In contrast, Maxwell and colleagues randomised 63 patients with trauma to receive APRV or LTV.[17] Acute Physiology and Chronic Health Evaluation II physiology scores were higher in those receiving APRV, but there were no statistically significant differences in either ventilator-free days or mortality between the groups.

Zhou and colleagues conducted a prospective RCT of 138 patients randomised to receive APRV or LTV.[18] At 28 days, the APRV group had spent less time undergoing mechanical ventilation {median ventilator-free days: 19 [inter-quartile range (IQR): 8–22] *vs* 2 (IQR: 0–15); $P<0.05$}, was more probable to be

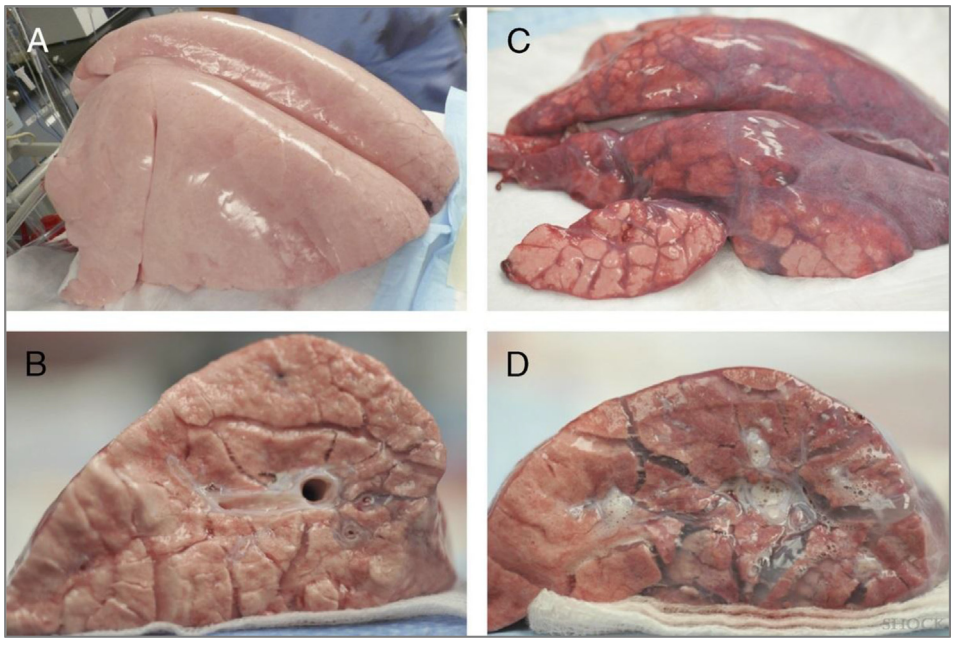

Fig 2 Macroscopic histological changes in a porcine model of airway pressure release ventilation (APRV) vs low tidal volume ventilation (LTV). This figure has been reproduced with permission from *Shock*. Gross pathology: (A) APRV whole lung – normal pink, homogenously inflated lung tissue with no evidence of inflammation or atelectasis. (B) APRV cut surface – no bronchial or septal oedema. (C) LTV whole lung – predominantly atelectatic with heterogenous parenchymal inflammation. (D) LTV cut surface – gel-like oedema seen filling the interlobular septae with airway oedema in the bronchial openings.

successfully extubated (66.2% *vs* 38.8%; $P<0.05$), and was less likely to need a tracheostomy ($P<0.05$). The APRV group had a significantly shorter stay in ICU [15 days (IQR: 8–21) *vs* 20 days (IQR: 10–32; $P<0.0\%$)], significantly better PF ratios, significantly better respiratory system compliance, but similar haemodynamic variables. In this trial, P-low was set at 5 cmH_2O in contrast to the method described by Habashi.[2] Whilst this study was not powered for this outcome, a non-statistically significant difference in ICU mortality was observed in the APRV group compared with the LTV group (19.7% *vs* 34.3%).[18]

A recent RCT comparing APRV and LTV in children was terminated early after enrolment of 58 children because of increased mortality in the APRV arm (53.8% *vs* 26.9%; $P=0.089$).[19] Notably, the severity of ARDS was greater in the intervention group, and a proportion of the children who died in the APRV group had been crossed over to LTV (28.5%) or escalated to high-frequency oscillatory ventilation (35.7%). High-frequency oscillatory ventilation is a technique that has subsequently fallen out of favour in adult practice, largely because of the increased mortality demonstrated in patients receiving this therapy in the Oscillation for Acute Respiratory Distress Syndrome Treated Early (OSCILLATE) trial.[20] The authors also acknowledged that, because of a specific pathology, a proportion of the patients in the intervention arm would not benefit from the alveolar recruitment offered by APRV.

Neurocritical care

Avoidance of hypoxaemia is a key component of neurocritical care and anaesthesia. Traumatic brain injury (TBI) or raised intracranial pressure (ICP) is often considered to be a relative contraindication to the use of APRV. Two retrospective case series and our own experience of using APRV in hypoxaemic patients with TBI and ICP monitoring already *in situ* have not shown a detrimental effect on ICP, and improved arterial oxygenation can often reduce a raised ICP.[21,22]

In addition to the avoidance of hypoxaemia, the maintenance of normocarbia is important when treating intracranial hypertension, and it may not be safe to tolerate permissive hypercarbia associated with a pH >7.2 in these patients. Our own practice is to note the minute volume with conventional ventilation required to maintain the target arterial carbon dioxide tension before switching to APRV, and then immediately titrate settings to maintain this minute volume. If this is impossible to achieve, ventilation is re-established with a more conventional mode and other methods of improving hypoxaemia are attempted. As at present there are no large data sets of the use of APRV in TBI, we would strongly recommend only using APRV when ICP monitoring is in place to allow determination of any positive or adverse effects on ICP and cerebral perfusion pressure.

Comparison of APRV and prone ventilation

The evidence base for the effect of the prone position on reducing mortality in moderate-to-severe ARDS is robust, with a multicentre RCT demonstrating a reduction in 28 day mortality from 32.8% to 16%.[23] As such, prone positioning should be a standard of care in all patients with ARDS and a PF ratio <150 mmHg. We would recommend this strategy before commencing APRV for severe hypoxaemia in ARDS where possible, and believe that APRV cannot be recommended as an alternative to the prone position in the management of such patients. Exceptions include patients in whom prone positioning is contraindicated, or where a trial of prone positioning has failed to improve gas exchange. Varpula and colleagues demonstrated that the two approaches are not

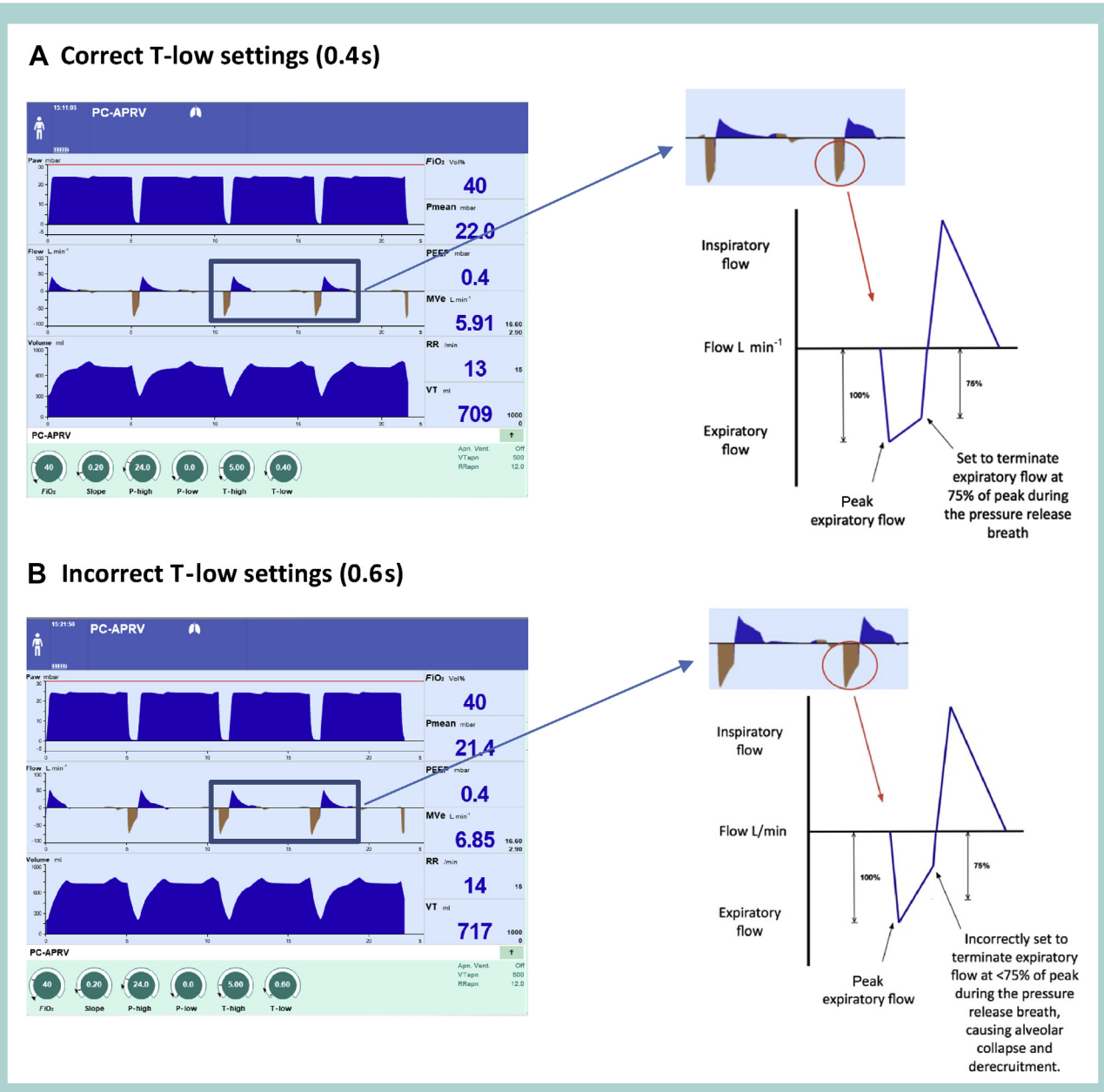

Fig 3 Adjustment of T-low to terminate expiratory flow at 75% of peak flow. Reproduced with permission from *Shock*. (A) Correct T-low settings (0.4 s). (B) Incorrect T-low settings (0.6 s).

mutually exclusive.[24] We occasionally initiate APRV in patients who are already in the prone position. This is achieved in a similar way to patients who are supine, but achieving spontaneous breathing in a patient in prone position can be challenging, and care must be taken to ensure a safe depth of sedation.

Initiating, titrating, troubleshooting and weaning from APRV

Initiating and titrating APRV

This section outlines a plan to initiating and titrating APRV. This approach, developed by Habashi, individualises ventilation settings for each patient.[2] Table 1 lists the indications and relative contraindications for APRV.

Ventilator manufacturers differ in the nomenclature used (Supplementary Table S2). They also vary in their delivery of the mode. In particular, APRV modes that add pressure support should have this feature removed, as this will continually adjust the duration of T-low automatically, which will potentially lead to airway collapse.[25] In addition, pressure support of spontaneous breaths when the lungs are inflated has the potential to cause barotrauma by over-distension of maximally recruited lung units. However, it is our experience that automatic tube compensation (ATC) is useful, and it should be used if available.

Table 1 Indications and contraindications for APRV

Indications	Contraindications (none are absolute)
Patients who are 'recruitable' (i.e. are considered to have collapsed/atelectactic areas of a lung)	Profound cardiovascular instability (particularly if secondary to untreated hypovolaemia)
Patients with diffuse disease process (e.g. ARDS and multifocal pneumonia)	Recent pulmonary resection with staple lines or anastomosis (i.e. postoperative lobectomy and pneumonectomy)
Patients in whom prone positioning is contraindicated or those who have not responded to a trial of prone positioning	Severe bronchospasm
Patients at high risk of respiratory deterioration (e.g. traumatic chest injury, aspiration, inhalational injury and pancreatitis)	Pulmonary hypertension with right ventricular decompensation (although a trial of APRV may be considered with close echocardiographic monitoring)
Patients with sepsis and multiple organ failure requiring invasive ventilation	Bronchopleural fistula
Obese patients	Untreated pneumothorax
Patients in whom a trial of low tidal volume ventilation has failed	Restrictive lung disease

Ventilator settings for the initiation of APRV will vary from patient to patient. Similar to the commencement of conventional mechanical ventilation, the settings will depend upon the condition of the patient and the preferences and experience of the clinician. A guide for the novice user is outlined in Box 1.

Most patients will already be receiving positive-pressure ventilation of some description, and those settings should be used to guide choice of initial APRV settings. All patients should already have a cuffed tracheal or tracheostomy tube *in situ*. They should also have invasive arterial monitoring *in situ* with rapid access to vasopressors and fluids. Ideally, patients should be sedated to a degree sufficient to tolerate the tracheal tube only and be breathing spontaneously. However, often, patients who may benefit from APRV are deeply sedated and receiving neuromuscular blocking drugs in an attempt to optimise their gas exchange. We advocate initiating APRV concurrently with cessation of neuromuscular blocking agents. Sedative drugs can then be weaned once monitoring has demonstrated no residual neuromuscular block. Sedation practices vary between ICUs, and the choice of sedative drugs should be tailored to each individual patient. An approach that uses drugs with a short duration of action can make titration and the promotion of spontaneous ventilation easier.

Patients have different and changing time constants and APRV settings should be individualised for each patient. Adjusting T-low to achieve expiratory flow termination at 75% of peak expiratory flow is vital for this mode to be effective and should be reassessed after any alterations to the ventilator. Setting the correct T-low maintains the correct end-expiratory volume for each patient, and failure to adjust it correctly may cause lung derecruitment.

The short T-low time ensures that the lungs never fully empty. This generates 'auto-PEEP', and therefore, despite setting P-low at 0 cmH_2O, intrathoracic pressure should never equalise with atmospheric pressure. Of note, it has recently been suggested that setting a P-low of 5 cmH_2O reduces driving pressure and may minimise derecruitment and atelectrauma, but this is not universally accepted.[18]

In patients receiving APRV, tidal volumes measured by the ventilator can exceed 6 ml kg^{-1} with the settings discussed previously. It is difficult to attempt to mirror ARDSnet tidal volume limitation, and this is not our usual practice.[4] We feel that applying one specific element of a mandatory mode of

Box 1
Initiating APRV.

(i) Set FI_{O_2} (fraction of inspired oxygen) to 1.0, but this can usually be reduced rapidly.
(ii) Set P-high to current plateau pressure. In reality, this is usually $\leq$30 cmH_2O), but increased pressures may be necessary in morbidly obese patients.
(iii) Set P-low to 0 cmH_2O.
(iv) Set T-high to 5 s (other values can be used [range: 3–8 s], but we start with this value for ease of recollection).
(v) Set T-low initially to 0.5 s (range: 0.3–0.8 s).
(vi) Check that T-low is short enough to terminate expiratory flow at approximately 75% of peak expiratory flow. This is done by examining the flow/time graphical display on the ventilator, as shown in Fig. 3(A). If the expiratory flow terminates at <75% of peak expiratory flow, as demonstrated in Fig. 3(B), shorten T-low by steps of 0.1 s until the flow terminates at 75%. Similarly, a flow trace indicating that expiratory flow terminates too early will need prolongation of T-low in steps of 0.1 s.
(vii) Stop neuromuscular blocking agents and gradually reduce sedative drugs to encourage spontaneous breathing. The use of shorter-acting sedative medications allows for easier titration.
(viii) Tolerate hypercapnia, aiming to maintain pH $\geq$7.25 as with conventional ventilation (with the exception of neurocritical care patients).

ventilation care bundle (ARDSnet) to a different paradigm of spontaneous ventilation (APRV), whilst well intentioned, is not rational. The limited animal and human trials conducted so far do not support titrating APRV to a particular millilitre per kilogram tidal volume target.

Permissive hypercapnia during mechanical ventilation is a strategy that has been widely adopted to facilitate the benefits of lung-protective ventilation. The degree of hypercapnia and respiratory acidosis tolerated by each patient will differ, and although we have recommended a value of pH $\geq$7.25, many patients tolerate further decreases in pH to $\geq$7.2. Conversely, other groups of patients will not tolerate even moderate degrees of hypercapnia, particularly in neurocritical care, those with coronary artery disease, congestive cardiac failure, arrhythmias, pulmonary hypertension, right ventricular dysfunction and significant hypovolaemia.

Troubleshooting with APRV

Measures useful in troubleshooting for a patient receiving APRV are outlined in Table 2. If gas exchange does not improve with these measures within the first 4–6 h of commencing APRV, it is our experience that the patient will probably not benefit.

Weaning from APRV

A scheme for weaning a patient from APRV is described in Box 2. It is important to remember that, as APRV encourages spontaneous breathing, it can be considered as a weaning mode in its own right.

Switching to conventional pressure assist mode of ventilation (if required)

We suggest the following:

(i) Remove ATC (if applicable).
(ii) Switch to PEEP of 12–15 cmH_2O with a pressure support of 10 cmH_2O.

Transfer of patients receiving APRV

The majority of patients undergoing APRV will be in the ICU. Whilst undertaking transfers of these patients for a scan or procedure, we recommend transferring whilst they are still

Box 2
Weaning APRV.

(i) Begin by reducing FI_{O_2}.
(ii) Once FI_{O_2} has reduced to 0.4–0.5, start to reduce P-high.
(iii) Reduce P-high by 2 cmH_2O every 2–6 h whilst maintaining FI_{O_2} at 0.4–0.5. If this causes hypoxaemia, increase P-high by 4 cmH_2O and wean more slowly.
(iv) Once P-high is 20 cmH_20, increase T-high by 1–2 s each time P-high is decreased. Continuing this will wean the patient to CPAP <10 cmH_20 with minimal releases, allowing a treating clinician to assess a patient's readiness for extubation.
(v) Alternatively, once P-high is 12–15 cmH_2O with FI_{O_2} 0.4, the patient can be switched to a conventional pressure supported mode, setting PEEP at the level of P-high with a low level of pressure support (e.g. 5 cmH_20) and weaned conventionally from there.

Table 2 Troubleshooting with APRV

Physiological derangement	Responses
Hyperoxaemia	(i) Reduce FI_{O_2} first. (ii) Once the FI_{O_2} is at 40–50%, start to reduce P-high.
Hypoxaemia	(i) Increase P-high by 2 cmH_2O. (ii) Increase T-high by 0.5–1 s. (iii) If T-high >10 s, consider reducing T-low by 0.2 s. (iv) Increase FI_{O_2}.
Hypercapnia	(i) Tolerate hypercapnia if pH >7.25 and there are no adverse effects of acidosis. (ii) Ensure that the patient is making spontaneous ventilatory effort and aim to reduce sedation further to enhance this. (iii) Ensure that ATC (if applicable) is set to 100% with the correct tracheal tube size. (iv) Decrease T-high by 0.2 s down to a minimum of 3 s. (v) Check that ventilator circuit and heat and moisture exchanger filter are free of secretions or excessive moisture. (vi) Consider increasing P-high to maximise recruitment and minimise dead space.
Hypocapnia (assuming adequate cardiac output)	(i) Increase T-high by 0.2 s. (ii) If oxygenation is adequate or excessive, decrease P-high.
Hypotension	(i) Administer a fluid bolus of 250–500 ml crystalloid. (ii) Adjust or initiate vasoactive medicines. (iii) Consider urgent echocardiography to assess filling status and biventricular function.

connected to the ICU ventilator if possible, or choosing a portable ventilator that can deliver APRV. Alternatively, a brief period of conventional ventilation may be considered, accepting the risk of derecruitment and subsequent hypoxia. It is important to keep the PEEP high (we suggest a minimum of 15 cmH_2O) when temporarily switching to conventional ventilation to minimise derecruitment.

Summary

When contemplating the heterogeneous nature of respiratory failure in intensive care, it is probable that one mode of ventilation does not provide optimum support for every patient with respect to gas exchange or survival. In this article, we have summarised the physiological rationale for and against the use of APRV, and the evidence to date supporting its use, and explained how APRV can be initiated, titrated and weaned.

Whilst APRV has an attractive theoretical basis, there are no large multicentre RCTs supporting its use, and the evidence base remains inconclusive with legitimate concerns about some of the adverse effects, notably in paediatrics. Future research should aim to clarify which specific subgroups of patients, if any, would benefit from the use of APRV, and whether APRV prevents the development of ARDS in at-risk patients. Despite the current paucity of good-quality evidence supporting its use, APRV is gaining in popularity within critical care. We believe APRV remains a useful tool in the armament of an experienced intensivist, particularly when caring for patients with SARF.

Declaration of interest

The authors declare that they have no conflicts of interest.

MCQs

The associated MCQs (to support CME/CPD activity) will be accessible at www.bjaed.org/cme/home by subscribers to *BJA Education*.

Supplementary material

Supplementary material to this article can be found online at https://doi.org/10.1016/j.bjae.2019.12.001.

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